MIT, intimal area (IA), lumen area (LA) and intimal index (II = IA/IA + LA). In the first year after heart transplantation, there was significant progression of disease in all patients. There was no difference in the progression of disease between group 1 and group 2 (see table). Regression analysis showed no relation between baseline TCAD and progression at one year for all measurements ($R^2 = 0.0$, $p = ns$).

	n	ΔIA (mm^2)	ΔMIT (mm)	ΔII
Group 1 (baseline MIT ≥ 0.3 mm)	17	1.2 ± 1.3	0.14 ± 0.12	0.062 ± 0.05
Group 2 (baseline MIT < 0.3 mm)	31	1.1 ± 1.2	0.15 ± 0.14	0.078 ± 0.09

Conclusion: pre-existing CAD does not appear to influence the progression of TCAD as assessed by morphometric analysis of ICUS in the first year after heart transplantation.

9:45

736-6 Two Year Follow-Up of the Functional and Morphologic Adaptation of Undersized Donor Hearts Following Cardiac Transplantation

Paul J. Mather, Valluvan Jeevanandam, Ileana L. Piña, Kenneth B. Margulies, Howard J. Eisen, Alfred A. Bove. *Temple University School of Medicine, Philadelphia, Pennsylvania*

We analyzed the ability for growth of 14 undersized, and 14 normal sized donor hearts over a period of two years after heart transplantation. Donor to recipient weight ratios were 0.53 ± 0.06 for small hearts, and 0.98 ± 0.05 for normal sized hearts. The left ventricular chamber sizes including the diastolic (LVd) diameter, and left ventricular mass(LVM) were obtained by M-mode and two-dimensional echocardiography. Measurements were obtained before procurement, at 10 weeks, and then at six month intervals post orthotopic heart transplantation.

	10 Weeks		One Year		Two Years	
	Undersized	Normal	Undersized	Normal	Undersized	Normal
LVM-gm	245±24	164±48*	240±32	156±40*	233±34	172±38*
LVd-cm	3.72±0.7	4.33±0.9	4.81±0.5	4.52±0.6	4.78±0.6	4.61±0.5
VO2	13.9±2.1	14.6±3.4	18.6±3.5†	20.1±2.9†	23±4.9†	24.9±4†
PVR	3.6±0.6	2.5±0.6	2.8±0.6	2.6±0.6	2.8±0.5	2.4±0.7

*$p < 0.05$ (comparing normal to undersized hearts), †$p < 0.05$ (within the groups), PVR = pulmonary vascular resistance (wood units), VO2 = oxygen consumption exercise test (ml/kg/min).

The increase in LVM, and LVd in the undersized hearts suggested that the left ventricle adapted to the larger recipient circulation over time. The functional capacity of the two groups were not significantly different. Despite denervation and a mismatched load, transplanted hearts adapt appropriately to their new hemodynamic milieu, thus suggesting the cardiac donor pool can be expanded to include undersized hearts.

737 Clinical Heart Rate Variability

Tuesday, March 26, 1996, 8:30 a.m.–10:00 a.m.
Orange County Convention Center, Room 414C

8:30

737-1 Different Patterns of Heart Rate Variability in Patients Suffering Sudden Death While Wearing Holter Monitoring: The Role of Concomitant ST Segment Changes

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Aim of the study was to investigate the relationship between sudden death and heart rate variability (HRV) in pts with ischemic heart disease (IHD). We analyzed the tapes of 10 pts with IHD suffering sudden death during Holter monitoring (HM): 6 pts (67 ± 7 yrs) with angina as well as "ischemic" sudden death (ISD: arrhythmic death preceded by ST shift), and 4 pts (70 ± 10 yrs) with ischemic cardiomyopathy but not ST changes before the fatal arrhythmia (ASD). Two matched groups of pts with IHD who underwent HM were used as controls: 18 pts with transient myocardial ischemia (G1), but not ventricular arrhythmias, were compared to ISD; 12 pts with non sustained ventricular tachycardia (G2), but not transient ischemia, were compared to ASD. Arrhythmias, ST segment and HRV were analysed by a computized interactive HM system. All sudden death pts showed ventricular tachyarrhythmias as their terminal event; pts with ISD showed ST depression starting 49 ± 36 min before sudden death. SDNN (sd of normal R-R intervals) in ISD was 102 ± 20 ms, as assessed by the overall 10 ± 7 hrs of HM, whereas it was 58 ± 15 ms and 31 ± 9 ms ($p < 0.01$ vs the initial 5 min time period), respectively 1 hr and 5 min before the onset of fatal ischemic ST shift. Comparison with G1 pts showed a significant difference only 5 min before ST shift (31 ± 9 vs 56 ± 30 ms: $p < 0.01$). In ASD pts SDNN during 9 ± 4 hrs of HM was 61 ± 21 ms ($p < 0.02$ vs ISD), whereas it measured 70 ± 37 ms and 43 ± 20 ms ($p = ns$), respectively 1 hr and 5 min before the onset of ventricular tachyarrhythmia. ASD group had lower SDNN values only during the overall HM period, when compared to G2 pts (130 ± 31 vs 61 ± 21 ms: $p = 0.001$). In conclusion: different patterns of sympathovagal imbalance may be found in pts with IHD suffering sudden death during HM. Indeed, an abrupt decrease of HRV may trigger fatal arrhythmias in ISD pts — i.e. with concomitant transient myocardial ischemia-, whereas pts suffering ASD show a low HRV during overall HM. Analysis of HRV indices could be useful to understand the role of autonomic activity in the occurrence of sudden death.

8:45

737-2 A Mathematical Model for Analyzing Sinus Arrhythmia

David P. Slovut, Richard B. Moeckel, John C. Wenstrom, Julie Floyd, Betty Hansen, Jerome H. Abrams. *University of Minnesota, Minneapolis, MN*

Objective: To quantify the respiratory component of sinus arrhythmia for innervated subjects and heart transplant recipients.

Methods: We recorded the respiratory cycle and EKG during spontaneous respiration for 10 healthy innervated subjects and 11 non-rejecting heart transplant recipients (denervated subjects). Each recording averaged 3.5 minutes. Interbeat (RR) intervals were normalized by subtracting the average heart rate per lung cycle from the instantaneous heart rate. Phase-response curves (PRC) comprised of lung phase versus normalized heart rate were constructed. Lung phase ranged from 0° (start-inspiration) to 360° (end-expiration). A trigonometric function was fitted to each PRC and used to assign an amplitude and phase to the sinus arrhythmia. Correlation coefficients were calculated to quantify the goodness of fit between the PRCs and the trigonometric curves.

Results: A high correlation was observed between PRCs and the trigonometric curves for innervated (μ r = 0.74, std = 0.13) and denervated (μ r = 0.75, std = 0.16) subjects. A t-test showed no significant difference in r value between groups. For both groups, inspiration was associated with an increased heart rate. In 9/11 transplant patients, the maximum heart rate occurred earlier in the respiratory cycle than for the innervated subjects (mean phase angle 106° vs 175°, $p < 0.001$). The remaining transplant patients showed decreased heart rate with inspiration. Innervated subjects showed markedly greater heart rate variability than transplant patients (mean 6.8 beats/min vs 0.87 beats/min, $p < 0.001$).

Conclusion: Healthy innervated subjects and heart transplant recipients demonstrate predictable, phasic changes in heart rate with respiration that can be well-approximated by a simple trigonometric function. The use of PRCs in characterizing normal cardiopulmonary interactions may improve our ability to diagnose cardiac dysfunction and cardiac allograft rejection noninvasively.

9:00

737-3 Heart Rate or Heart Rate Variability for Risk Stratification After Myocardial Infarction?

Xavier Copie, Katerina Hnatkova, Ann Staunton, Lu Fei, A. John Camm, Marek Malik. *St George's Hospital Medical School, London, UK*

Heart rate variability (HRV) is a powerful tool for risk stratification after myocardial infarction. Although, HRV is related to heart rate, little is known of the predictive value of heart rate after myocardial infarction.

Methods: We compared the power of left ventricular ejection fraction (LVEF), HRV, and mean RR interval for the prediction of all-cause mortality, cardiac mortality, and sudden cardiac death in 579 patients after myocardial infarction. Mean RR interval and HRV index were computed from predischarge 24-hour Holter recordings.

Results: During two years of follow-up, there were 54 deaths, 42 of which were cardiac (26 sudden). Shorter mean RR interval was a better predictor of all-cause mortality, as well as cardiac and sudden death than depressed LVEF. Depressed HRV predicted the risk of death better than mean RR interval for sensitivities below 40%. For sensitivities above 40%, mean RR interval was as powerful as HRV. For cardiac death prediction, a LVEF < 35% had a 40% sensitivity and 14% positive predictive accuracy, HRV < 17 units had a 40% sensitivity and 20% positive predictive accuracy, and a